Case Report
Colorectal tubular adenoma bleeding in patients with decompensated cirrhosis—a case report

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Abstract: Background: Esophageal variceal bleeding (EVB) is one of the most common complications in patients with decompensated cirrhosis, and there is rare of patients with decompensated cirrhosis complicated by intestinal bleeding due to benign tumors, which is prone to misdiagnosis. In November 2016, our hospital diagnosed and treated 1 case of liver cirrhosis patient with colorectal tubular adenoma bleeding. Case presentation: A 65-year old male was admitted to our hospital in November third, 2016, due to repeated haematemesis more than 1 year and blood in the stool for 4 hours before admission. One years ago, he was hospitalized in our hospital diagnosed as hepatitis B decompensated cirrhosis, due to vomiting blood and discharging black pulpy stool. The patient was underwent endoscopic variceal ligation and postoperative recovery was good. The patient was discharged with regular medication (propranolol 10 mg, 2 times/day; entecavir 0.5 mg, 1 time/day; esomeprazole 20 mg, 2 times/day), and 6 preventive treatments of esophageal variceal ligation in previous hospitalization. The patient achieved excellent therapeutic effect because of positive endoscopic treatment of suspicious lesions.

Keywords: Esophageal variceal bleeding, decompensated cirrhosis, sigmoid colon tumor, gastroscopy, colonoscopy

Background
Esophageal variceal bleeding (EVB) is one of the most common complications in patients with decompensated cirrhosis. The common manifestations include hematemesis and melena or bloody stool. Hematemesis and melena often appear sequentially, but there are some patients who only have melena or bloody stools, and no hematemesis. However, there is rare of patients with decompensated cirrhosis complicated by intestinal bleeding due to benign tumors, which is prone to misdiagnosis. In November 2016, our hospital diagnosed and treated 1 case of liver cirrhosis patient with colorectal tubular adenoma bleeding.

Case presentation
A 65-year old male was admitted to our hospital in November third, 2016, due to repeated haematemesis more than 1 year and blood in the stool for 4 hours before admission. One years ago, he was hospitalized in our hospital diagnosed as hepatitis B decompensated cirrhosis, due to vomiting blood and discharging black pulpy stool. The patient was underwent endoscopic variceal ligation in our hospital and postoperative recovery was good. The patient was discharged with regular medication (propranolol 10 mg, 2 times/day; entecavir 0.5 mg, 1 time/day; esomeprazole 20 mg, 2 times/day), and 6 preventive treatments of esophageal variceal ligation. The last time was March 1st, 2016. Three months ago, the patient was treated in our hospital due to cerebral infarction and improved after treatment. After oral administration of aspirin “100 mg QD” treatment, the patient was discharged with haemoglobin (HGB) examination 122 g/L.
Four hours before admission, the patient began to discharge dark red blood stool, a total of 5 times about 800 ml, not mixed with the stool, no hematemesis, no abdominal pain, and no syncope. In another hospital, the patient showed no improvement by stopping taking aspirin after examination of HGB (72 g/L) and treating with esomeprazole (8 mg/h intravenous injection), octreotide (50 µg/h, 24 h intravenous injection with micro pump maintenance) and other drugs, and blood transfusion (O type Rh positive red suspension 4U). Therefore, the patient was transferred to our hospital to continue treatment.

Physical examination after admission showed that temperature was 36°C; pulse was 115 times/min; breathing was 21 times/min; blood pressure was 95/62 mmHg; conjunctiva and lips were slightly pale; there were no yellow sclera and conjunctival edema; lungs was clear without dry and wet rales; heart rate was 115 beats/min; each heart valve auscultation area was no noise. The abdomen was soft without tenderness and rebound tenderness, and liver and spleen were not palpable under ribs. Bowel sounds was active with 12 times/min and much dark red bloody fluid was visible in anus. Finger examination of anaus was negative and no edema was visible in both lower extremities.

Blood routine examination after admission showed that white blood cell count was 5.26×10⁹/L; HGB was 85 g/L; platelet count was 112×10⁹/L. Prothrombin time (PT) were 11.4 seconds, the international normalized ratio (INR) was 0.96, activated partial thromboplastin (APTT) was 34.4 seconds and the total amount of fibrinogen was 3.42 g/L. After admission, the patient discharged 2 times of dark red blood stool about a total volume of 200 ml. Patients admitted to hospital was diagnosed as gastrointestinal bleeding.

Patients had a history of cirrhosis with EVB, but the patient discharged dark red stool without hematemesis performance. Esophagogastroduodenoscopy (EGD) is a reliable method for diagnosis of EVB within 12 to 24 hours of bleeding [1]. Emergency gastroscopy after hemosta-
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The tumor was treated by sigmoid colon tumor root ligation treatment (Figure 4) and biopsy.

Postoperative daily monitoring of blood showed that HGB increased progressively (HGB 95 g/L after 3 days). Pulse was 85 beats/min and blood pressure was 135/70 mmHg. Colonoscopy after oral purgative bowel preparation showed that terminal ileum, ileocecal, ascending colon, transverse colon, descending colon and rectal mucosa showed no abnormality. Sigmoid colon tumor after root ligation showed no active bleeding. Pathological biopsy showed tubular adenoma (sigmoid colon). One week after admission, the HGB was recovered to 110 g/L. The occult blood of stool was negative. The patient was discharged. One month after discharge, colonoscopy review showed that there was no residual tumor in sigmoid colon (Figure 5), and there was no obvious abnormality in the terminal ileum and large intestine mucosa.

Discussion

Hepatitis B virus is the main cause of cirrhosis in China. Esophageal varices (EV) are a common complication of cirrhosis. The main performances are hematemesis and melena. Patients are at a high risk of bleeding and death after stop of the acute variceal bleeding. For patients without preventive treatment, the average recurrence rate in 1 to 2 years was 60% and the mortality rate was 33% [2]. So the prevention is very important including drug therapy, endoscopic therapy, surgery or radiation therapy [3-5]. Non selective beta receptor blockers (commonly used drugs propranolol) can reduce the risk of bleeding and improve the survival rate [6, 7]. Endoscopic variceal ligation (EVL) was used for the prevention of EV rebleeding [8, 9]. A number of clinical studies demonstrated that non selective beta receptor blockers combined with endoscopic therapy is the first choice for prevention of EVB [10, 11]. The patient was given propranolol combined with endoscopic variceal ligation (a total of 7 times) after initial esophageal varices. According to our clinical experience, the esophageal varices should be controlled. But it must be given clear diagnosis as soon as possible to determine whether it was EBV, because misdiagnosis might be life-threatening. With gastroscopy, we proved that our initial diagnosis at admission was not EVB.
Polyps of colon include adenoma, hyperplastic polyps and hamartoma [12]. Adenomas can be divided into tubular, tubular villous and villous adenoma. The tubular adenoma, with a tubular structure and the villi component less than 20%, is one of the most common gastrointestinal polyps composed of hyperplastic mucosa epithelium. Endoscopic manifestations show multiple or single nodular surface. Most are pedunculated and the size is generally not more than 2 cm. They are dark red and easy to bleed. Under a microscope, they are hyperplasia of glandular tissue. Glandular epithelium was arranged regularly and well differentiated, with atypia, much mitosis, but not invading the muscle membrane. In tubular adenoma, large volume and wide base tumors with nodular or lobulated surface and surface erosion and hemorrhage are easy for canceration. A report showed that [13], the canceration rate of adenomatous polyps with the diameter of 1~2 cm is about 10% and the diameter more than 2 cm is close to 50%. Therefore, the adenomatous polyps should be treated as soon as possible. Endoscopic high-frequency electroblation resection [14] is the preferred method of treatment of colon polyps, but has the higher incidence of hemorrhage and perforation to remove wide base and thick pedicle polyps [15]. Bleeding is the most common and most serious complication with the incidence rate is about 0.3%-6.1% [16, 17] and the perforation rate is about 0.085%-0.18% [18, 19]. Nylon rope ligation can reduce the risk of bleeding and perforation [20].

At present, low-dose aspirin (75~150 mg/D) is widely used in the treatment of coronary atherosclerotic heart disease (CHD), cerebrovascular disease and peripheral arterial disease [21]. Studies have shown that aspirin can increase the risk of gastrointestinal injury 2 to 4 times [22]. Meta analysis showed that the absolute risk of severe gastrointestinal bleeding caused by aspirin was 0.12% per year and was associated with doses [23]. Primary prevention meta-analysis showed that aspirin increased the incidence of gastrointestinal bleeding by 1.37 times [24]. The mechanism of gastrointestinal bleeding because of [25, 26]. 1) local effects: aspirin has a direct stimulating effect on the phospholipid layer on the gastric mucosa, destroying hydrophobic protective barrier of gastric mucosa; the disintegration of aspirin in stomach induced a large amount of release of leukotrienes, damaging gastric intestinal mucosa and 2) systemic effects: aspirin can induce serine acetylation in the active site of cyclooxygenase (COX), inhibiting the activities of COX-1 and COX-2 in gastric mucosa, and decreasing the production of prostaglandin. PG regulates gastrointestinal blood flow and mucosal function and its reduction is the main cause of gastrointestinal mucosal injury caused by aspirin. In recent years, the incidence of lower gastrointestinal bleeding was significantly higher in patients receiving dual antiplatelet therapy and most patients combined with PPI [27]. It usually occurs within 12 months after taking the drug and the peak reaches [28] at 3 months. The patient was characterized by a clear history of decompensated cirrhosis with EVB and was given 3 months of medication of aspirin. But he was admitted because of hematochezia without vomiting and hematemesis. Lowering portal pressure and acid suppression therapy in another hospital failed to effectively stop bleeding and EVB diagnosis was not clear. Under the premise of maintaining the stable life signs of the patient, emergency gastroscopy was performed to exclude the possibility of EVB and upper gastrointestinal bleeding. The first colonoscopy found suspicious bleeding lesions (sigmoid colon tumor: tubular adenoma), and treated by endoscopic snare ligation; second underwent colonoscopy identified the cause of the bleeding and third colonoscopy identified the complete removal of sigmoid colon tubular adenoma.

Conclusions

In this case, through clinical observation and analysis, EVB was excluded by gastroscopy and lower gastrointestinal bleeding due to sigmoid colon tumor was diagnosed by colonoscopy. The patient achieved excellent therapeutic effect because of positive endoscopic treatment of suspicious lesions.

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Disclosure of conflict of interest

None.

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